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## Categorising myocardial infarction with advanced cardiovascular imaging

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A 66-year-old man collapsed at home with chest pain. He was found by paramedics to be in ventricular fibrillation leading to a cardiac arrest. A post-resuscitation electrocardiogram showed sinus rhythm and inferolateral ST-segment elevation. On arrival at our department he had an emergency invasive coronary angiogram which showed an occluded left circumflex artery, confirming myocardial infarction (MI). We opened the artery using a balloon, and following recanalisation, we identified a substantial filling defect indicative of a large thrombus (figure). Following stent implantation, normal coronary artery flow was restored and the patient made an uneventful recovery—although brief paroxysms of atrial fibrillation were noted on cardiac monitoring in the ensuing 24 h.

The patient enrolled in a research study (NCT03943966) of PET/CT angiography using a novel radiotracer,  $^{18}\text{F}$ -GP1, which binds to activated platelet glycoprotein IIb/IIIa receptors and provides an assessment of thrombus formation. CT angiography confirmed a patent stent that co-localised with intense  $^{18}\text{F}$ -GP1 uptake on the PET/CT images, consistent with acute thrombus trapped behind the stent (figure).

Incomplete contrast opacification in the left atrial appendage made assessment on CT challenging. However, the presence of a separate and discrete filling defect raised the possibility of an in-situ appendage thrombus, which was confirmed on the  $^{18}\text{F}$ -GP1 PET/CT scan (figure). The clinical diagnosis was therefore changed from type 1 MI—caused by an atherosclerotic plaque rupture—to a type 2 MI—caused by a coronary embolism arising from a left atrial appendage thrombus in the context of paroxysmal atrial fibrillation.

The patient was commenced on apixaban 5 mg twice a day, aspirin 75 mg daily, and clopidogrel 75 mg daily. The patient was allowed home after 2 days and made a full recovery.

Differentiation between type 1 and type 2 MIs is important because different therapeutic interventions are required to prevent recurrence. Most MIs—type 1—are due to acute coronary occlusion from a thrombus forming on a ruptured atherosclerotic plaque. In

type 2, MIs are caused by a mismatch between the supply and demand of myocardial oxygen in the context of another disease—including coronary embolism—as seen in our patient. Adjunctive invasive or non-invasive imaging can often help clarify the clinical diagnosis and guide management (appendix; video).

### Contributors

We all provided care for the patient and managed the case. We were all involved in drafting and writing the final manuscript. Written consent for publication was obtained from the patient.

### Declaration of interests

We declare no competing interests.

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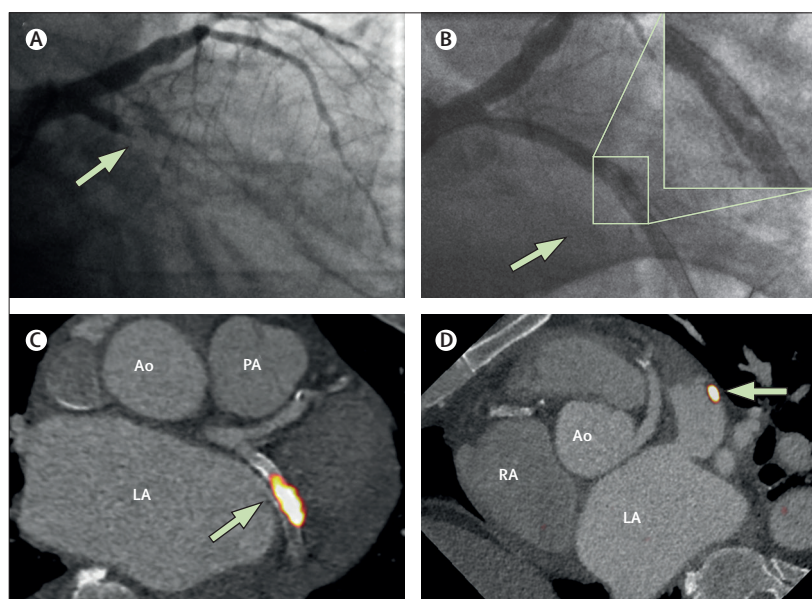
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See [Online](#) for appendix

See [Online](#) for video



**Figure: Typing a myocardial infarct**

Invasive coronary angiography shows a proximally occluded left circumflex artery (A; arrow) and a persistent filling defect following recanalisation (B; arrow).  $^{18}\text{F}$ -GP1 PET/CT shows uptake co-localised to the stented coronary segment indicating thrombus behind the stent (C; arrow).  $^{18}\text{F}$ -GP1 PET/CT scan shows uptake co-localised to a filling defect indicating an additional thrombus in the left atrial appendage (D; arrow). Ao=aorta. RA=right atrium. LA=left atrium. PA=pulmonary artery.